Remarks

In the Office Action dated July 16, 2003, claims 30-36, in the above-identified U.S. patent application were rejected. Reconsideration of the rejections is respectfully requested in view of the above amendments and the following remarks. Claims 30-31 and 33-36 remain in this application, claims 1-29 and 32 have been canceled.

Claims 30-36 were rejected under 35 USC §112, first paragraph. The office action indicates that fragments containing only the CDR3 region are not enabled. Applicants respectfully disagree with this contention but in order to advance the prosecution of the present application, the claims have been amended to recite the sequences for the CDR1 and CDR2 regions. In addition, the claimed antibodies and/or antigen binding fragments have already been shown to have therapeutic applicability (see Example 3 of the present application) and the phagemide clones identified in Example 1 are Fab antibody fragments. Therefore, the present application does include specific disclosure regarding antibody fragments. In view of the above amendments and comments, applicants request that this rejection be withdrawn.

Claims 30-36 were rejected under 35 USC §102(b) as anticipated by Berchtold. Applicants respectfully point out that Berchtold does not disclose antibodies with the claimed sequences. It is well known that all mammals are capable of generating several billion different antibody specificities. Different antibody specificities are also generated against the same antigens because the majority of antigens (such as GPIIb/IIIa) contain a number of different epitopes. In view of this, it is highly unlikely that Berchtold's antibodies would have the claimed sequences particularly since the antibodies in Berchtold were obtained from diseased patients.

Claims 30-36 were rejected under 35 USC §102(b) as being anticipated by Nugent. Nugent et al discloses a human monoclonal IgM autoantibody binding to GPIIIa with increased avidity on aging platelets. In contrast thereto, the claimed antibodies are derived from a human monoclonal IgG antibody and they bind to the GPIIb/IIIa complex. Dissociation of the GPIIb/IIIa complex by EDTA results in loss of binding of antibodies. The above claim amendments clarify this issue. The claimed antibodies do not bind to GpIIb alone and they inhibit binding of fibrinogen to human GPIIb/IIIa (a fibrinogen binding test is described in Example 3 and in Figure 4). In view of the above amendments and comments, applicants request that this rejection be withdrawn.

Claims 30-36 were rejected under 35 USC §112, second paragraph as indefinite. The claims have been amended to clarify that the antibody is an isolated human antibody which is able to bind to GPIIb/IIIa wherein the heavy chain of the antibody comprises CDR1, CDR2 and CDR3 regions with the recited sequences. Claim 35 has been amended to clarify that the antibody according to claim 30 is in combination with a second human antibody. Claim 31 has been amended to indicate that the antibody includes the variable domain of the Lchain of a human antibody. In view of these amendments, applicants request that these rejections be withdrawn.

In the event this paper is not considered to be timely filed, the Applicant respectfully petitions for an appropriate extension of time. Any fee for such an extension together with any additional fees that may be due with respect to this paper, may be charged to Counsel's Deposit Account No. 02-2135.

RESPECTFULLY SUBMITTED					
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